AMENDMENTS TO THE SPECIFICATION:

Please add the following new paragraph after the title, on page 1:

This application is a divisional application of Application No. 09/674,973, which was PCT filed on May 3, 1999.

Please amend the paragraph beginning at page 8, line 29 and ending at page 9 line 18, as follows:

--In our International Application PCT/NO92/00032 (published as WO92/14756), the content of which is herein incorporated by reference, we described synthetic peptides and fragments of oncogene protein products which have a point of mutation or translocations as compared to their proto-oncogene or tumour suppressor gene protein. These peptides correspond to, completely cover or are fragments of the processed oncogene protein fragment or tumour suppressor gene fragment as presented by cancer cells or other antigen presenting cells, and are presented as a HLA-peptide complex by at least one allele in every individual. These peptides were also shown to induce specific T cell responses to the actual oncogene protein fragment produced by the cell by processing and presented in the HLA molecule. In particular, we described peptides derived from the P21 *ras* protein which had point mutations at particular amino acid positions, namely position 12, 13 and 61. These peptides have been shown to be effective in regulating the growth of cancer cells *in vitro*. Furthermore, the peptides were shown to elicit CD4+ T cell immunity against cancer cells harbouring the mutated P21 *ras* oncogene protein through the administration of such peptides in vaccination or cancer therapy schemes. Later we have shown that these peptides also elicit CD8+ T cell immunity

against cancer cells harbouring the mutated P21 *ras* oncogene protein through the administration mentioned above.--

Please amend the paragraph beginning at page 39, line 33 and ending at page 40 line 10, as follows:

--Below are listed some examples of where these mutations may result in gene products that result in development of tumours:
Development of colorectal cancers are believed to result from a series of genetic alterations. Deleted in colorectal cancer (DCC) gene (seq id nos 30-34), Human cysteine protease (ICErel-III) gene (seq id nos 394-398 and 459), Human putative mismatch repair/binding protein (hMSH3) gene (Seq id nos 134-147), Human hMSH6 gene (seq id nos ~~201-204~~ 200-203 and ~~295-299~~ 293-297, Human n-myc gene (seq id nos ~~190-195~~ 189-194), Human TGFβ2 (hTGFβ2) gene (seq id nos 22-29), Human p53 associated gene (seq id nos ~~287-294~~ 285-292 may be involved in colorectal cancer.--

Please amend the paragraph beginning at page 40, line 12 and ending at line 16, as follows:

--Human breast cancer susceptibility (BRCA2) (seq id nos 35-94) and Human BRCA1-associated RING domain protein (BARD1) gene (seq id nos ~~404-413~~ 404-417) are involved in breast cancer and ovarian cancer Human hMSH6 gene (seq id nos ~~201-204~~ 200-203 and ~~295-299~~ 293-297) may be involved in brain tumours.--

Please amend the paragraph beginning at page 40, line 22 and ending at line 34 as follows:

--Human breast cancer susceptibility (BRCA2) gene (seq id nos 35-94), Deleted in colorectal cancer (DCC) gene (seq id nos 30-34), Human putatative mismatch repair/binding protein (hMSH3) gene (seq id nos 134-147), Human hMSH6 gene (seq id nos ~~201-204~~ 200-203 and ~~295-299~~ 293-297), human N-MYC gene (seq id no ~~190-195~~ 189-194), Human TGFb2 (hTGFb2) gene (seq id nos 22-29), Human p53 associated gene (seq id nos ~~287-294~~ 285-292), Human MUC1 gene (seq id nos ~~248-267~~ 247-266), Human germline n-myc gene (seq id nos ~~184-195~~ 182-188), Human Wilm's tumour (WIT-1) associated protein (seq id nos 388-393), Human nasopharynx carcinoma EBV BNLF-1 gene (seq id nos ~~205-211~~ 204-210), Human transforming growth factor-beta ~~inducted~~ induced gene product (BIGH3) (seq id nos ~~228-233~~ 227-232).--

Please amend the paragraph beginning at page 41, line 1 and ending at line 6, as follows:

--Many of the mutated genes may result in development of leukemias and lymphomas: Human neurofibromin (NF1) gene (seq id nos ~~178-183~~ 176-181), b-raf oncogene (seq id nos ~~172-177~~ 170-175), Human protein-tyrosine kinase (JAK1) gene (seq id nos ~~268-272~~ 267-271), Human protein-tyrosine kinase (JAK3) gene (seq id nos ~~273-280~~ 272-279) are examples.--

Please amend the paragraph beginning at page 41, line 8 and ending at line 12, as follows:

--Genes involved in malignant melanoma: Human malignant melanoma metastasis-supressor (hKiSS-1) gene (seq id nos ~~331-337~~ 328-334), Genes involved in metastasis: Human metastasis-associated mtal (hMTA1) gene (seq id nos ~~360-365~~ 357-362).--

Please amend the paragraph beginning at page 41, line 14 and ending at line 31, as follows:

--Cell cycle control and signal transduction is ~~strikcly~~ strictly regulated. Frameshift mutations in these genes may result in uncontrolled cell growth. Examples of genes which may be ~~susceptable~~ susceptible are: Human protein tyrosine phosphatase (hPTP) gene (seq id nos 95-102), Human kinase (TTK) gene (seq id nos ~~109-121~~ 109-120), Human transcriptional repressor (CTCF) gene (seq id nos ~~122-128~~ 121-127), Human cell cycle regulatory protein (E1A-binding protein) p300 gene (seq id nos ~~212-219~~ 211-218), Human transforming growth factor-beta ~~inducted~~ induced gene product (BIGH3) (seq id nos ~~228-233~~ 227-232), Human FLt4 gene (for transmembrane tyrosinase kinase (seq id nos ~~281-286~~ 280-284), Human G protein-coupled receptor (hGPR1) gene (seq id nos ~~317-322~~ 314-319), Human transcription factor (hITF-2) gene (seq id nos ~~329-330~~ 326-327), Human telomerase-associated protein TP-1 (hTP-1) gene (seq id nos ~~338-351~~ 335-348), Human transcription factor TFIIB 90 kDa subunit (hTFBIIB90) gene (seq id nos ~~366-373~~ 363-369), Human FADD-homologous ICE/CED-3like protease gene (seq id nos ~~129-133~~ 128-133)--

Please amend the paragraph beginning at page 41, line 33 and ending at page 42 line 2, as follows:

--Mutations in DNA synthesis or ~~repair~~ repair enzymes may also lead to uncontrolled cell growth. Human DNA topoisomerase II (top2) gene (seq id nos 103-108) and Human putative mismatch repair/binding protein (hMSH3) gene (seq id nos 134-147) and (hMSH6) gene (seq id nos ~~201-204~~ 200-203 and ~~205-299~~ 293-297).--

Please amend the paragraph beginning at page 42, line 4 and ending at line 11, as follows:

--The following are tumour suppressor genes, Human retinoblastoma binding protein 1 isoform I (hRBP1) gene (seq id nos ~~148-158~~ 148-156), Human neurofibromin (NF1) gene (seq id nos ~~178-183~~ 176-181), Human p53 associated gene (seq id nos ~~287-294~~ 285-292, Human retinoblastoma related protein (p107) gene (seq id nos ~~312-316~~ 310-313), Human tumour suppressor (hLUCA-1) gene (seq id nos ~~374-381~~ 370-377), Mutations in these genes may result in development of cancer.--

Please amend the paragraph beginning at page 42, line 13 and ending at line 20, as follows:

--The following are oncogenes, proto-oncogenes or putative oncogenes; Human germline n-myc gene (seq id nos ~~184-189~~ 182-188), Human n-myc gene (seq id nos ~~190-195~~ 189-194), Human can (hCAN) gene (seq id nos ~~300-302~~ 298-300), Human dek (hDEK) gene (seq id nos ~~309-311~~ 307-309), b-raf oncogene (seq id nos ~~172-177~~ 170-175), Human DBL

(hDBL) proto-oncogene/Human MCF2PO (hMCF2PO) gene (seq id nos ~~303-308~~ 301-306).

Frameshift mutations in these genes may lead to development of cancer.--